THE CAUSES OF DEATH IN DIPHTHERIA,

WITH

NOTES ON TREATMENT.

E. MANSEL SYMPSON, M.D.

1891.

Reprinted from THE PRACTITIONER.]

THE CAUSES OF DEATH IN DIPHTHERIA, WITH NOTES ON TREATMENT.

BY E. MANSEL SYMPSON, M.A., M.D., B.C. CAMB.,

Lincoln.

IN the following pages by diphtheria is meant an acute specific contagious disease, characterised by the formation of false membrane [1] on some portion of the respiratory tract, of the other mucous membranes, or of the skin, by great constitutional depression and anæmia, by albuminuria, and occasionally by peculiar after-paralyses.[2]

As a basis for this paper the records of eighty cases have been taken from two wards, one male and one female, of St. Bartholomew's Hospital, by the kind permission of Dr. Gee. They are all the cases of diphtheria admitted to those wards for the past ten years; thus they may fairly well represent the general run of diphtheria in any large town. Thirty-seven out of the eighty were fatal; in forty-four the larynx and other respiratory passages were affected, in forty-two false membrane was seen on the fauces, in nine it was coughed up or seen *post mortem* in the larynx or bronchi, while in thirteen it occurred both on the fauces and in the larynx; the total number of cases with false membrane being therefore sixty-four. Albuminuria was present in thirty-five cases, and nine were attacked by the special paralyses. Thirty-five were

[1] In epidemics catarrhal diphtheria may occur wherein there is no formation of false membrane whatever. These cases may communicate diphtheria to others and may be followed by paralysis.

[2] As in other diseases, one or more of these symptoms may be absent.

children below the age of six, and but little difference as to incidence was manifested between any of the quarters of the year, nearly the same numbers being noted in each.

In the following pages an attempt is made to follow out as clearly as possible all the causes of death in diphtheria to their ultimate factors, and to illustrate each one by a well-marked case.

In considering this subject, first in possible time of occurrence, though fortunately of great rarity in England, comes death due to *malignant diphtheria*, the primary blood-poisoning form.

None of the cases analysed at all correspond with this heading, so as an example one of two cases contributed by Sir Dyce Duckworth to the *St. Bartholomew's Hospital Reports*[1] will be taken.

A lad, aged 4½ years, who had not been very ill till June 28, then had had headache and vomiting, was admitted on the 29th. He looked ill and pale, his breath was offensive, the glands at the angle of his jaw were swollen, and there was a brawny collar from ear to ear. Very fœtid watery blood was discharged from the nostrils, on the soft palate were some shreds of friable membrane, and there were some purpuric patches on his legs. His temperature was never higher than 99·4. On the 30th he was more anæmic, and the odour from his nose and mouth was gangrenous. He became unconscious, and died quietly the same evening. *Post mortem*, the fauces were covered with shreds of brown gangrenous membrane, and both tonsils were ulcerated and gangrenous. The larynx and trachea contained shreds of brown dirty-looking membrane adherent to their walls, and one bronchus was plugged with membrane. The heart was natural. There were many patches of hæmorrhage in his lungs, and some in his pleuræ; the kidneys were natural and the spleen was not enlarged.

Clinically, in the quickly fatal termination, and the utter oppression of the patient, these cases bear a close resemblance to the adynamic form of *scarlatina maligna*. In this, as described by Dr. Gee, there is great depression, vomiting, great anxiety, and feeble delirium. The face becomes pale livid, and perfect stupor (interrupted by convulsions) supervenes, the pulse is excessively weak and frequent, respiration remarkably irregular, sweats break out, the skin is cold and mottled: these symptoms precede death, which may take place in twelve hours from the first seizure. The paralysis of the heart has been proved by the thermometer *not* to be due to excessive temperature.[2]

[1] Vol. xxiii. p. 15 *et seq.*

[2] *A System of Medicine*, J. Russell Reynolds, vol. i. p. 342.

But the hæmorrhagic tendency so evident in the case quoted invites comparison with another disease of blood-poisoning—septic intoxication or sapræmia. Here, in the acutest cases,[1] symptoms of collapse quickly set in. The pulse is feeble, rapid, and irregular, the temperature falls, and may even sink below normal. Consciousness is lost, and the patient may become comatose before death. Dyspnœa is a common symptom on the last day, and the urine frequently contains albumen. *Post mortem*, the heart will be found flabby, with sub-pericardial petechiæ, and similar extravasations beneath the pleuræ and peritoneum. The lungs will display hypostatic congestion, the liver and kidneys will be swollen and full of blood, and the spleen swollen and soft.

Malignant diphtheria then partakes of the nature of malignant scarlatina and of sapræmia, and death therein results from failure of the heart's action. How the poison acts on the heart will be considered later on.

Next in order of time comes death by *syncope*. This may take place at any period of the disease, and the danger of its occurrence does not cease even after the false membrane has definitely vanished, if diphtherial paralysis has ensued.

The two following cases illustrate quite sufficiently what is only too common a history in this disease:

Annie R., aged 2, on December 9, 1886, was sick and languid (an older child died in the same house a week earlier of diphtheria), and had some discharge from the nose and mouth. On December 10 there was swelling under both ears, slight dyspnœa, and her voice changed. She was admitted on December 13, when the notes are as follows:—Fairly good colour, glands behind angles of jaw are large and hard. There is no membrane visible on the fauces. Chest—some sibilus over the bases of both lungs, but there is no recession or dyspnœa. Urine, sp. gr. 1020, with one-tenth of albumen. December 14—temp. 100°, respir. 42, urine sp. gr. 1030, with one-half of albumen. Membrane on uvula (this was afterwards removed, it fitted like a finger-stall) and right tonsil. Colour pallid, breath very offensive, and there is great discharge from the nose. December 15—worse, cough brassy, though she has no dyspnœa, temp. 100·4°. December 16—died quietly.

The second case is that of Mary C., aged 5. She was admitted on September 11, 1885. For a week she had had a lump in her neck and pain on swallowing, and on September 10 she lost her voice. On the 11th her colour was very pale, the glands at the angles of her jaw and the superficial cervical glands were swollen. There was some nasal discharge, and a slough was seen on the soft palate. On

[1] Erichsen's *Science and Art of Surgery* (8th edition), vol. i. p. 943.

September 12 the urine was half albumen. On September 13 there was no laryngeal trouble or dyspnœa, and the child died quite quietly on suddenly sitting up in bed.

Both these patients, it will be observed, had severe albuminuria—an evidence, I think, of the profound constitutional disturbance—and both suffered from nasal diphtheria, which indeed might place them under the heading of secondary septicæmia. That is one danger of the nasal affection, *i.e.*, the increased difficulty of treatment; another, as Dr. Squire[1] remarks, is that suckling children are liable to perish from starvation, as they cannot suck when the inflammation blocks up both nostrils. And it seems reasonable to think that the diphtherial poison gets a chance of entering more readily and in larger quantity into the system by way of the blood when the false membrane is situated on such a highly vascular (especially venous) mucous membrane as that of the nares, than when it is seated in the fauces, larynx, or trachea. These two cases are specially selected as devoid of complications, and it must be evident that any addition, such as even a slight attack of bronchitis or laryngitis, would—combined with this tendency to heart-failure—overcome the patient speedily.

Thus a tendency to syncope has probably carried off several of the cases whose notes have been analysed, who would perhaps have made a successful fight with a simple laryngitis or pneumonia, and although it holds no high place among the statistics of mortality, indirectly it is very largely responsible for the largeness of that mortality. As with malignant diphtheria, the cause of syncope will be considered in treating of diphtherial paralysis.

Of this paralysis a brief sketch may make clear the symptoms and pathology of fatal cases occurring during its course.

The palsy generally comes on in convalescence, *i.e.* after the throat has recovered, from two to six weeks from the beginning of the illness. In *four* of my cases it was noticed at the end of the *first* week, in *two* at the end of the *second*, in *one* after a *month*, and in the remaining *two* after the lapse of *six weeks*.

The seat of this special form of paralysis is in the cranial and

[1] Reynolds's *System*, vol. i. p. 402.

spinal nerves. Of the former, those chiefly and most generally attacked are the *third*, as shown by the loss of accommodation and sometimes by the paralysis of one or more of the ocular muscles supplied by this nerve; the *spinal accessory* and the *glossopharyngeal*, as shown by the palsied palate, and the *vagus*. whereby the action of the larynx, pharynx, and heart is interfered with or abolished. The *spinal* nerves which supply the trunk-muscles and the extremities suffer, as the loss of deep reflexes, the paresis or actual paralysis of the muscles, and in some instances the affections of sensation, testify.

The order of occurrence is that the palate or pharynx is usually affected first, in other instances the legs and arms. And it is very interesting to note that even in cases of cutaneous diphtheria (where there has been no membrane at all on the fauces) the palatal palsy has appeared. And the paralysis is said to appear first—in cutaneous diphtheria—in the muscles adjacent to the lesion of the skin.

Now morbid anatomy shows tolerably clearly that the chief feature of diphtherial paralysis is a neuritis—peripheral or multiple—of certain nerve-cords. Sometimes, too, there is some disease evident of the anterior horns of grey matter in the spinal cord, and occasionally degenerative changes are found in the palsied muscles—changes which are almost certainly secondary to the disease of the nerves.

Thus this seems to be a special form of paralysis, coming on after the action of diphtheria in the skin or mucous membranes is over, it is transitory (rarely lasting more than three or four months), and it attacks certain nerves much more frequently than others. It can be compared then fairly accurately with the paralysis produced by alcohol or lead. Both these poisons select (if the term be allowed) special nerves (nay, in the case of lead a special portion of one nerve), giving rise to peculiar palsies which at first (*i.e.* if the cause be removed early enough) are transitory in character.

It is submitted that there is good reason for thinking that the diphtherial poison—when it enters the patient's body in an extreme dose—kills him outright (with the symptoms of malignant diphtheria) by paralysing the heart, by its action on the vagus, in exactly the same way as an over-dose of nicotine

would do. In the heart-failure of blood-poisoning there certainly are two ways at least whereby the weakness of the heart can come about; either the special poison acts on the nervous apparatus of the heart, or the state of the blood is so altered for nutrition that the muscular fibre of the heart gets feebler and feebler, and a time comes when it refuses to act at all—the heart stopping full of blood in diastole. The latter condition is probably the predominant cause of the asthenia of sapræmia: both possibly have some, but I think the former far the larger share, in causing stoppage of the heart in malignant diphtheria.[1] And the *ante-mortem* clots which observers have found in the chambers of the heart in cases of diphtherial syncope are doubtless due to the weakened action of the heart, and would themselves serve as a fresh source of embarrassment.

Also, when smaller doses of the poison are taken (as in uncomplicated cases such as have been related above), a gradual depression of the heart's action takes place, due likewise to the action of the poison on the vagus, and death occurs by syncope. And, as would be the case in nicotine-poisoning, whether death were brought about by one fatal dose or by several smaller ones, no appreciable pathological change has been found in the nerves in either the malignant or the more ordinary cases of syncope in diphtheria. Later on in the disease, indeed, in special persons (just as in lead- or alcohol-poisoning), but, as it is contended, simply as a *continuation* of the same process, this irritation of the vagi and other nerves produces definite traces of its presence by the inflammation of their branches. Lead-poisoning affects generally nerves whose *rôle* is not the immediate preservation of life; luckily perhaps the same remark will apply to alcohol-poisoning in its more chronic forms, but when, as with diphtheria, a poison acts almost directly on the heart, it is obvious that many must get killed by it who would otherwise have suffered from diphtherial paralysis; in other words, that personal idiosyncrasy acts as much in determining the immediate or remote effects of a dose, as it does in making the person susceptible to the disease at all. And it is noteworthy in this

[1] Though Oertel says, "In the most severe forms of septicæmia and poisoning the blood is slightly coagulable, sticky, brown, or rather livid, and soils the fingers like sepia."—*Ziemssen's Cyclopædia*, vol. i., p. 647.

connexion that the paralysis is more common in adults, and in those who have been hard worked mentally and bodily.

A fatty change has indeed been described in the heart-muscle by Dr. Bristowe, Dr. Hillier, and Dr. Mosler[1]; this perchance may have been due to a primary affection of the nerves supplying the heart. But this condition is evidently rare, and deaths from syncope are comparatively common in diphtheria. And in no way (save by their earlier occurrence and the absence of any paralytic symptoms) do these deaths from syncope differ from those proved to be due to a definite inflammation of the vagus.

The causes of death resulting from neuritis of the glosso-pharyngeal, spinal accessory, vagus, and spinal nerves may be now considered.

The palsy of the palate by itself does not harm the patient much, except by not shutting off the posterior nares while swallowing is proceeding, hence it allows part of his food to return through the nose.

Paralysis of the pharynx *is* a serious matter, especially if the epiglottis be palsied and insensitive, as then the power of swallowing may be greatly diminished or almost entirely lost, food may get into the larynx and cause possibly fatal obstruction, or into the lungs and set up pneumonia.

Paralysis of the pharynx is also serious, as it interferes with the process of feeding children by a tube—through the nose into the stomach—which may get caught in the palsied pharynx and pour out its contents there to overflow into the larynx and trachea.

Paralysis of the glottis is full of danger, in the case of any bronchial effusion or the spread of membrane down into the bronchial tubes, as, from the larynx not being under control, coughing to expel the contents of the tubes is often quite impossible, or so weak as to be ineffectual. And, when the glottis is paralysed, and the power of separating the vocal cords absent—after the operation of tracheotomy has been performed—

[1] Dr. Bristowe's case is in the *Pathological Transactions*, vol. x., and is near akin apparently to that quoted from Sir Dyce Duckworth. Dr. Hillier mentioned two cases in his book on *Diseases of Children*, p. 140. Dr. Mosler's name is given on the authority of Senator, *German Clinical Lectures*, Second Series, New Sydenham Society, p. 422.

the tracheal tube cannot be taken out, and the wound be permitted to close.

As a rule, in neuritis of the vagus, its branches are attacked, not the nerve as a whole.

The symptoms of inflammation of its cardiac branches are as follows: The pulse may be greatly increased in frequency, or gradually reduced to extreme slowness, and I have noted in these cases that the first sound of the heart is scarcely audible. In both states there may be irregularity, though this by itself is not uncommon in the convalescent stage of all acute diseases. Suddenly, as the patient undergoes some slight exertion, sits up in bed, or uses the bed pan, the heart ceases to act. Or, in the more gradual form, the force and frequency of the heart-beats diminish, till life seems to flicker out like a wasted candle. A case[1] related by Dr. Ransom of Cambridge illustrates this kind of death, I think, exactly.

A girl of fourteen whose throat had been well (after an attack of diphtheria) for several days, and who seemed to be rapidly recovering, had some nausea and vomiting one evening followed by slight epigastric pain and dyspnœa. Death occurred the next morning, the eighteenth day, quite suddenly, apparently from syncope. Here, the age of the patient, the late period of the seizure, the vomiting, dyspnœa, and sudden death, point I think indisputably to neuritis of the branches of the vagus supplying the heart, lungs, and stomach.

Inflammation of the gastric branches reveals itself by persistent vomiting and by that alone. Now vomiting in diphtheria[2] may be due to actual deposit of the false membrane in the stomach, whether it has been infected by pieces swallowed, or whether it has spread down the œsophagus by continuity: but this is very rare.

Or it may be the result of uræmia, as not infrequently there is a good deal of albuminuria in these cases, and the late Dr. Hillier gives one[3] which bears out this point. Or it may be a

[1] *Brit. Med. Journal*, 1887, vol. ii., p. 1387.

[2] For much of this section on diphtherial vomiting I am indebted to a clinical lecture given by Dr. Gee, which he has most kindly allowed me to use. I may refer here also to an article by Dr. Gee in the twenty-fifth volume of the *St. Bartholomew's Hospital Reports* on the same subject.

[3] *Op. cit.* p. 139.

solitary symptom of diphtherial paralysis, coming on earlier than the more common ones. The two following cases will furnish illustrations of death from persistent vomiting.

Emma H., aged 9, was admitted July 18, died July 29. A sister had just died in another ward. On July 13, she had a sore throat and some difficulty in swallowing. Condition on admission : neck swollen, a muco-purulent discharge from her nose, false membrane on the fauces, slight dyspnœa, voice natural, no laryngeal symptoms. Urine sp. gr. 1030, much (about one-third) albumen. During the next few days the patient improved considerably, the membrane cleared off the throat, the swelling of the neck subsided, the urine became less albuminous (on July 24 the coagulated deposit was only one-sixth of the urine), and the patient seemed to be doing well until July 26 when the vomiting began. On July 28 it was noted that the patellar reflex was absent, and that the vomiting was incessant ; this continued so till her death. The *post-mortem* examination showed no noteworthy signs of disease except in the kidneys.

Again, Emily T., aged 3½, was admitted June 29 and died July 7. A sister died in this ward from diphtheria on June 27, and on that day patient had a sore throat. Condition on admission : pale, with a muco-purulent discharge from the nose, membrane on the fauces, glands enlarged at the angles of the jaw. July 2. Urine albuminous. July 4. Vomiting greatly ; breathing noisy, but not stridulous. The vomiting persisted till death.

In the first case the vomiting commenced on the eighth day ; in the second on the fifth day of the disease. Among Dr. Gee's cases, its commencement took place once on the third day, once on the sixth, once on the seventh, twice on the eighth, twice on the tenth day, and once after the lapse of three weeks. In both the cases quoted, and in several of Dr. Gee's, there was albuminuria, and of course the vomiting might be uræmic. But in the first case given, and in Nos. VIII. and IX. of Dr. Gee's, there was distinct evidence of the co-existence of diphtherial paralysis, and the condition of the urine was improving before vomiting set in. And, as we shall see later on, numbers of patients have some affection of the kidneys during diphtheria, but very rarely do they give any signs of this save by passing albuminous urine. So that it seems probable that vomiting coming on in a persistent way may be due to a neuritis of the vagus-branches to the stomach, just as syncope occurring early without other paralytic symptoms may be due to a neuritis of the cardiac branches. But on these points more *post-mortem* evidence of the condition of the nerve-branches and trunks is greatly to be desired. These, then, are all the affections of the vagus likely to cause death.

But it is not so very uncommon to get palsy of the diaphragm, resulting from neuritis of the *phrenic* nerves. I have notes of a well-marked case of alcoholic multiple neuritis, in which for more than ten days before death the diaphragm had ceased to act at all, there was no protrusion of the abdomen, or descent of the viscera during inspiration. Just so in diphtheria, although none of the cases here analysed exhibited this feature.

Two of Dr. Caiger's cases,[1] both fatal, show the condition alluded to very clearly. One was that of a man, aged twenty-five, who had pharyngeal diphtheria. The membrane persisted, he became unable to swallow, the patellar reflexes disappeared, the diaphragm acted very feebly, and he became cyanosed. The second was that of a boy, aged five, suffering from faucial diphtheria. Here, too, the membrane persisted, he had some dyspnœa, ocular palsy, absence of patellar reflex, and inaction of the diaphragm. The danger to life, when there is even slight bronchitis, apart from the tendency to syncope, is obviously great.[2]

The intercostal nerves may be affected, producing corresponding paralysis of the intercostal muscles, but fortunately this palsy and that last-mentioned very seldom are at their worst simultaneously.

A case [3] narrated by Dr. Abercrombie exemplifies this form.

A lad, aged 7, had diphtheria with some albuminuria. On the sixteenth day (when the membrane had gone and the urine was free from albumen) he was convulsed, and afterwards had right frontal headache and left facial paralysis. On the seventeenth day he was again convulsed, and this was followed by complete left hemiplegia. Naso-pharyngeal palsy supervened, and he died on the twenty-eighth day from paralysis of the intercostals. *Post-mortem*, the right middle cerebral artery was found to be occluded at its bifurcation by a firm, pale, adherent thrombus.

This case also introduces the subject of paralysis, other than diphtherial, occurring during the course of or after diphtheria. In children hemiplegia, sometimes paraplegia, comes on

[1] *Lancet*, Dec. 14, 1889, p. 1222.

[2] Also see a case of the late Sir W. Gull's in the *Lancet* for 1858, vol. ii. p. 5, where the breathing became entirely thoracic. The diaphragm was unmoved in inspiration, and depressed in expiration, indicating a loss of power in the phrenic nerves; and another of Dr. G. W. Rachel's in the *Archives of Pediatrics*, Sept. 1890, p. 686.

[3] *International Medical Congress*, London, 1881, vol. iv. p. 64.

suddenly after any of the acute febrile diseases such as measles, scarlatina, or diphtheria. On this point Dr. Ashby has raised the question whether these attacks may not be the result of extremely high temperature ; to settle the question, obviously more clinical observations are required. Dr. Abercrombie's case did not die directly from the hemiplegia, but Dr. Gowers [1] quotes two fatal cases out of three of Mendel's, in one of which a minute hæmorrhage, the size of a cherry-stone, had damaged the internal capsule. Henoch [2] also records a case as follows. Left hemiplegia occurred during the stage of collapse in diphtheria. The cause revealed by the *post-mortem* examination was the formation of a thrombus in the left appendix auriculæ, and an embolism in the Sylvian artery which had proceeded from it. Wallenberg also mentions three cases (out of 160) of this kind. Professor Humphry [3] records one of sudden right hemiplegia about a fortnight after the beginning of diphtheria. The patient was practically choked by the mucus in his lungs, and at the necropsy there was found a small suppurated spot, with softening of the adjacent brain-substance, in the superficial part of the left cerebral hemisphere.

The acute nephritis of febrile infectious diseases such as diphtheria is likened by Cohnheim to the effects of cantharides upon the kidney.

Pathologically, there are two kinds of diphtherial nephritis, (1) a glomerulo-nephritis, and (2) an acute disseminated (interstitial) nephritis, accompanied by great infarction of the stellate veins.[4] It is seldom a serious affection, dropsy rarely occurs, nor does the attack—unless in a very exceptional case—lead on to the chronic forms of kidney-disease, whereof scarlatinal nephritis is so frequently a commencement.[5] As we have seen

[1] *Diseases of the Nervous System*, vol. ii. p. 835.

[2] *Diseases of Children*, New Syd. Soc. translation, vol. i. p. 273.

[3] *Brit. Med. Journal*, 1863, vol. ii. p. 4.

[4] Fürbringer (*Practitioner*, vol. xxxi. p. 300) gives three classes—

I. Parenchymatous degeneration of the epithelium in the cortical tubules.

II. More intense degeneration spreading to the epithelial coverings of the glomeruli.

III. The large yellow kidney, with extensive degeneration of the parenchyma, and well marked degeneration of stroma.

[5] See Hilton Fagge's remarks on Oertel's cases of diphtherial dropsy, *Principles and Practice of Medicine*, vol. i. p. 286.

vomiting to a dangerous or fatal extent may be due to the nephritis. And as examples of death in this disease, entirely due to the nephritis, the two following cases will serve.[1]

A child of 5 years old, who died on the thirteenth day from the beginning of his diphtheria with dropsy and uræmic convulsions. After death, parenchymatous nephritis was all that could be found to explain it.

A child of 3, who had the false membrane on the throat and vulva. She died on the twentieth day from albuminuria and convulsions; parenchymatous and glomerulo-nephritis, and fatty degeneration of the heart, were found *post-mortem*.[2]

Now we can proceed to the most numerous class of deaths, those due to affection of the larynx, trachea and lungs. With regard to that battle-field of medicine—membranous croup—but little need be said here. If we differentiate off *laryngismus stridulus* (with its close association with rickets, its carpo-pedal contractions, and other evidences of its nervous origin); *spasmodic croup* (with its rapid onset and departure and tendency to recur); *catarrhal laryngitis* (the analogue, I believe, in children to the *œdema glottidis* of adults); and unmistakable *diphtheritic laryngitis*—all of which have answered to the term croup—we shall find only the so-called membranous croup left. Now in measles not rarely there is a membranous affection of the throat which may spread to the larynx and trachea. Also in the course of scarlet fever, the symptomatic sore throat becomes occasionally covered with false membrane, exactly the same in its characters and structure as that produced by the action of diphtheria; but as Senator[3] points out, this formation of membrane very rarely indeed spreads to the larynx and other air passages.

Membranous laryngitis then (dropping the term *croup*, which should only be used to denote a *symptom* common to many diseases, and should never be used *pathologically* to denote a disease, any more than "sore throat" should be) is a disease characterised by the development of membrane in the larynx and trachea, non-infective and non-contagious, not prostrating the system, except through the dyspnœa, accompanied by no

[1] Dr. J. Cassel, *Lancet*, 1890, vol. i. p. 36.

[2] Also see two cases of Drs. Ashby and Thompson, *Brit. Med. Journal*, 1890, vol. i. p. 962.

[3] *Op. cit.* p. 435.

albuminuria, and followed by no special paralysis. Death, if not due to the laryngeal asphyxia, may occur from extension of the membrane into the bronchi, from bronchitis, or from broncho-pneumonia. Henoch [1] gives records of some cases which fulfil these conditions, but judging from the report of the Medico-Chirurgical Committee, the discussion thereon, reports of cases since, and my own experience, I do not think idiopathic membranous laryngitis at all common in this country. I once admitted a small girl into a surgical ward, who had swallowed some boiling water from a kettle. She had typical membrane on her tonsils and uvula, stridulous breathing, croupy cough, and a metallic tone of voice. There was some recession of the chest during inspiration in Harrison's sulcus, and in the supra-clavicular spaces. She soon got rid of the membrane in the throat, coughed up some from the larynx, and quickly recovered. That *was* membranous laryngitis, though it owned a traumatic origin.

Again, may catarrhal laryngitis ever be diphtherial? Although false membrane is *per se* the sign of diphtheria, yet as mentioned above, we may get in epidemics a catarrhal affection of the fauces indisputably diphtherial, but stopping short of the formation of the diphtheritic false membrane; so we may probably get a catarrhal laryngitis only—but indisputably diphtherial—which does not develop any membrane whatever. In the cases analysed for this paper there was generally but little doubt as to their being genuinely diphtherial in their origin.

Five of them died of asphyxia from the presence of membrane in the larynx and trachea, twenty-one from its extension into the bronchi, four from bronchitis, and one from secondary pneumonia. In all but two of these cases tracheotomy was performed.

First, then, come the cases where the membrane has scarcely extended further than the larynx; the following is a good example:—

Drusilla S., aged 2, was sick on February 23, 1887, her voice changed and she began to cough. On February 24 the condition on admission was as follows—Colour good, no membrane visible, croupy cough and voice. Chest-sounds

[1] *Op. cit.* p. 370, *et seq.*

normal, some recession with inspiration in Harrison's sulcus and in the supra-clavicular spaces during inspiration. Pulse 120, temperature 100·4°, respirations 40. Urine free from albumen.

February 25, breathing more laboured, lips blue, face pallid, recession of chest more marked, pulse very feeble, 120, respirations 36 ; died in the afternoon.

Post-mortem, there was false membrane over the epiglottis extending right down to 1½ inches below the vocal cords, closely attached above and quite loose below them. The right lung was œdematous and engorged.

Next we come to cases where the membrane has occupied the trachea ; take the following as an example :—

Alice C., aged 4, had a cough and lost her voice on December 7, 1885. On admission, December 9, she was pale blue, there was much dyspnœa, and the glands at the angles of her lower jaw were much swollen. At 1.40 P.M. tracheotomy was performed, the child died on the table, and a long cast of false membrane was removed from the trachea.

Extension of the false membrane into the lungs is *the* most fatal form of diphtheria, and it is the chief factor in heightening the melancholy statistics of tracheotomy. As far as this kind is concerned indeed, we can hope for no better results from intubation of the larynx. An example including every point of interest is the following—

Henry W., aged 3, had a swollen neck on September 11, 1885 (there was a history of recent diphtheria in his family). Admitted on September 14, when he was pale, the glands behind the angles of his jaw were swollen, and there was false membrane on the right tonsil, right arch of palate, and the uvula. Cough not croupy, and there was no dyspnœa.

On the 15th his voice was noticeably husky, and there was albumen in his urine.

September 16, metallic cough, supra-clavicular recession, *râles* all over chest. At 9 P.M. tracheotomy was performed.

On the 17th at 3.30 P.M. he died of pulmonary asphyxia. After death, the false membrane was seen to have extended from the fauces down into the larynx, trachea, and larger bronchi. The main bronchus on one side was almost stopped up with creamy stuff, while that on the other was only just patent.

Here then were all the symptoms of diphtheria ; the membrane on the fauces, the albuminuria, the anæmia, the glandular affection, and, besides, a history of infection. Four days after the beginning of his illness the membrane spreads from his throat into his lungs and kills him. Most of the cases which I have classed under this heading are much the same ; in some

there is not quite so much evidence for diphtheria as their cause, but the case quoted will sufficiently illustrate the general features of all.

As might be expected, inflammation of the bronchial tubes is not uncommon, and will be again mentioned when the dangers of tracheotomy are briefly recounted. The irritation of diphtheria, indeed, is quite enough often to set up bronchitis, apart from the influence of a wound and a tube in the windpipe. Both sources of irritation were present in the following case—

William C., aged 2½, seemed short of breath on January 2, 1884. On admission on the 4th, there was false membrane on the fauces and uvula and nasal discharge; tracheotomy was performed as there was great dypsnœa. He died on January 6, and both lungs were found to be choked with mucus.

Of pneumonia, secondary to diphtheria, I have only one case among the number analysed.

D. W., aged 2½ in June 1883, had a sore throat; on the 5th she was admitted, when there was membrane on the fauces, and the urine was albuminous. Tracheotomy was performed on the 6th, and membrane was expelled from the wound. The patient died on June 29, and the lungs were found to be in a state of pneumonic consolidation.

But diphtheria may also apparently travel in the opposite direction, from the lungs into the throat.

A few months ago, a lad, aged 14, under my father's care, was attacked by a somewhat acute bronchitis. On the third day, towards evening, his breathing became more laboured, and continued so with slight intervals of improvement all the night. There had not yet been any membrane on the fauces. On the morning of the *fourth* day, he had rapid stridulous respiration, almost complete aphonia, and supra-clavicular recession. His colour was livid, and scarcely any air entered his lungs, wherein were only a few *râles* to be heard. There was a small patch of membrane on the left tonsil. As there was no change for the better in a few hours' time, I performed tracheotomy, and introduced a Durham's silver cannula with great relief to his breathing. During the operation some false membrane was removed from the trachea with the dilator. For more than twenty-four hours he improved greatly, then his breathing became shallow and laboured, his temperature rose to 104°, and he died—thirty-seven hours after the opening of his trachea—from asphyxia, and although up to the moment of his death there was a full current of air passing in and out through the tube, no air seemed to enter his lungs. *Post-mortem*, there was false membrane extending from the larynx right down through the trachea and bronchi, and ending in mucus in the smallest tubes.

Here the bronchitis may have had nothing to do with the

diphtheria; or, as it certainly seemed at the time, it may have been the first effects of the attack. There was found to be some rather uncertain history of infection.

The *dangers* to life from tracheotomy scarcely perhaps come within the scope of a purely medical paper, but a short account thereof will add to its completeness.

The immediate dangers are as follows. *Death from shock*, as in the case of A. F., aged 2, who was admitted with stridulous dry cough, much recession of chest-walls, but no evident membrane. She died on the table. In another case, Walter B., twice or thrice during the operation he seemed to have gone, he stopped breathing and his radial pulse was hardly to be felt. This danger helps to complicate the question as to the advisability of giving chloroform for tracheotomy.

Hæmorrhage, which, so far as my experience goes, comes from the veins, and generally stops when the trachea is opened and the accumulation of blood in the lungs and right side of the heart has been relieved; hæmorrhage may be dangerous not so much from its amount as from the risk of its occurring into the trachea, and so suffocating the patient.

Of course in the process of opening the trachea the œsophagus may be wounded, but with due care such accidents should be avoided.

Among the remoter dangers of tracheotomy comes *extension of the diphtheritic process* to the edges of the wound, sloughing of the wound, and death from exhaustion therefrom. Again, the irritation of the tube in the trachea may set up a *chronic inflammation* around, giving rise to much dyspnœa, or spreading downwards and producing bronchitis, or the tube may ulcerate through the back wall of the larynx into the œsophagus. But the greatest danger of all occurs *when the tube cannot be removed*, whether, as mentioned above, from paralysis of the larynx, or from stenosis of the larynx, in the case of a child. For, sooner or later, there may come a time when the tube will be pulled out, by the patient or the patient's friends, and suffocation will speedily follow, unless some trained hand is near to replace it. A little girl about four years old, who had worn a tracheotomy

tube for nearly two years, was brought dead to me, as house-physician for the week at St. Bartholomew's. When she left the Hospital, she had been placed in a Home where she could be well looked after and where she was very happy. In spite of much advice and most stringent warnings, the parents took her home. The melancholy sequel came to pass, she was left alone for half an hour, pulled the tube out of her throat, and was suffocated.

A few notes on the *treatment* of diphtheria will very naturally follow on, and give a more practical value to, the conclusions arrived at previously in considering the various causes of death. But, before entering on the principles or practice of treatment, one point must be discussed—the essential nature of the diphtherial virus. On *a priori* grounds, this disease has been classed among the specific febrile ones, from its contagious and infective character, from the comparative definiteness of its symptoms, and the sameness of its course. More and more, as our knowledge increases, are such diseases referred to a particulate virus, to the action of certain vegetable organisms, bacilli or micrococci. Now the membranous formation, so often mentioned above, is the most characteristic and the most common feature in diphtheria. It seems to be the seat of contagion; from it particles are constantly given off, either in the form of sputa, or, when the throat is dry, perhaps in very small flakes, or almost dust. So that it was not very surprising that Loeffler discovered in this membrane bacilli which he considered peculiar to diphtheria. Two kinds of micro-organisms are indeed found in this membrane. First, multitudes of micrococci, which fill the lymph-channels leading from the affected part, and which are also found in the neighbouring glands. There seems to be nothing of a specific nature about these micrococci, nor do they give rise to any symptoms resembling those of diphtheria when injected into animals. Secondly, bacilli of a particular kind appear in the false membrane, and in the false membrane alone. They are about as long as the bacilli of tubercle, but twice as thick, and they have not yet been noticed in any of the internal organs of the human body. When introduced into animals,

they give rise to symptoms comparable with those of diphtheria in man. MM. Roux and Yersin[1] have confirmed these results. They find that typical false membranes can be produced by inoculation of the cultures on abraded mucous membranes in rabbits and guinea-pigs, and that an injection under the skin causes œdema of the part and death. The pathological appearances *post mortem* vary with the animal, but no multiplication of the bacillus in the tissues takes place, so that death results from poisoning from the neighbourhood of the inoculation-wound. Here, evidently, should be a useful distinction between the membrane of diphtheria and that of membranous laryngitis and pharyngitis, whether idiopathic, or due to scarlatina or measles, wherein various forms of micrococci may be present, but not the diphtheria bacillus. And this distinction is not a mere fancy one, useful only for theorising, for the authors (MM. Roux and Yersin) give full directions for examining the membrane.

From clinical experience then, as well as from the observations quoted above, it seems that the action of the diphtherial virus is entirely local, that the germs of the disease do not themselves affect or attack the general system of the body, and probably as a result of this we find that one attack of diphtheria does not protect against another. Then we must regard the false membrane as the product and the dwelling-place of micro-organisms, which there manufacture a poison to be sent into the blood certainly once and probably very frequently. In the earlier part of this paper the action of this poison has been explained and illustrated. MM. Roux and Yersin have been successful in isolating a special chemical substance which when injected into animals gives rise to almost the same symptoms as the injection of the bacillus itself, with one exception—that no false membrane is formed. This is quite what we should expect, if we believe that the local irritation of the bacillus causes the formation of the false membrane.

Two questions then arise naturally at this point, as to the treatment of the false membrane, and as to the general treatment with reference to the poison which has been already taken into the patient's system. These two questions apply to the

[1] *Brit. Med. Journal*, 1890, vol. ii. p. 751.

treatment of all cases of diphtheria, in which there is any false membrane which can be seen or manipulated. Bretonneau and Trousseau generally tore off the membrane, and then painted the surface thus cleared with hydrochloric acid and honey. Latterly, the membrane has not been torn away, chiefly on the ground that not infrequently it speedily re-forms, that in children it is not very easy to do, and that it lurks in places (such as behind the epiglottis, &c.) which cannot be got at. Also MM. Roux and Yersin, laying stress on the extreme activity of the poison, say that if time has been allowed the bacillus to form a sufficient dose of poison, it is useless to remove the diphtheritic membrane. But this should not prevent some attempt being made to check the activity of the bacillus, for no one knows when the fatal limit may be reached. The best plan of treatment, then, would seem to be to swab the false membrane well with some fluid, or dust it with a powder, which will attack the bacilli, stop the formation of false membrane, and which will not harm the patient. For the proud position of being *the* fluid or powder which can act thus, some dozens of candidates, vegetable, mineral, acid, and alkali, are in the field of the medical press. So that I shall only mention one or two whereof I have had special experience. A combination of glycerine and carbolic acid, as in the *British Pharmacopœia*, is very useful for this purpose; both are antiseptic, as MM. Vidal and Chantemesse [1] found, and prevent the development of the bacilli, and the glycerine gives a stickiness to the compound which makes it stay about the throat for some time after application. They recommend five parts of carbolic acid to twenty-five of glycerine, with the addition of twenty parts of camphor. For mild cases, the acid might be replaced by boric acid or boroglyceride. The throat should be painted two or three times daily, or more if the case is very severe. Another agent which has given me excellent results in the membranous formations of scarlet fever is *tinctura ferri perchloridi*. It soaks into the membrane, and very soon the membrane comes away, leaving a clean healthy surface exposed. And Dr. Barraclough [2] has had

[1] *Brit. Med. Journal*, 1889, ii. 1484.
[2] *Lancet*, Jan. 25, 1890.

great success with the stronger tincture in the treatment of diphtheritic pharyngitis, for swabbing the false membrane. I have generally also seen a spray of carbolic acid (1 in 60—80) used frequently on the throat; but I think that for this lime-water might be substituted, as it is a powerful agent in dissolving the membrane. There are, as pointed out before, special dangers attending *nasal* diphtheria, as the membrane is formed on a very vascular mucous membrane, and so the patient is exposed to a more speedy and more abundant poisoning than is the case where the membrane is on the tonsils, uvula, and fauces generally. There is also the danger, if the membrane decomposes, of ordinary septicæmia occurring, and the nasal passages are much less easy to deal with than the throat. The nares may have a weak solution of *tinctura ferri perchloridi* syringed through them, or Dobell's solution, which I have used with great benefit in cases of ozæna and otorrhœa, may be substituted for it. If there is any threatening of septicæmia, a lotion of sulpho-carbolate of zinc will be of great service, syringed thoroughly through the nose while the patient breathes by his mouth. Full doses of salicylate of quinine, a most valuable drug for all septicæmic troubles, should be given. All this will be very hard to manage with children, in whom diphtheria of the nose usually is met with; but at any cost of time and patience the nose must be kept as free as possible from membrane and its decomposition. One method of clearing the nose is by making the child sneeze, by the use of snuff, and this might be made of salicylic acid.

General Treatment.—The chief dangers of diphtheria, apart from its killing through asphyxia, due to its spread to the larynx, trachea, and bronchi, are, as has been pointed out earlier in this essay, the occurrence of syncope at any period in the disease, and of paralysis of vital organs after the membrane has disappeared. In but few cases does the fever cause much trouble; from my experience, indeed, the thermometer helps but little in this complaint. From the very first, then, I would give small doses of iron, the citrate of iron and ammonium, or of iron and quinine, being the most convenient form when given alone. But, if digitalis is given—one of the very best preven-

tives of syncope—the *liquor ferri magnetico-phosphatis* is much the nicest and most elegant preparation, though too expensive for hospital patients. The digitalis will have no bad effect on the kidneys, rather the other way, for it will flush them and so prevent the poison lodging there and irritating them. And digitalis will be utterly free from risk as regards exciting any of the nervous elements liable to be attacked by the poison. This last reason is a slight objection to the routine use of *tincture of nux vomica* or *liquor strychninæ*, though both are good substitutes and are invaluable in the stages of paralysis. If any drug is at all an antidote to the poison of diphtheria, probably strychnine is that drug, and injected hypodermically and frequently in the worst cases it may tide a life over a dangerous crisis. Certainly, I have seen persons, apparently moribund from different diseases, kept alive till they took, as the popular term is, "a turn" and recovered. Of *chlorate of potassium* I have no very high opinion; it may easily be given to children to a dangerous extent, and its action on the kidneys too is not at all beneficial. It seems also to have very little effect on the mucous membranes in diphtheria when used in lozenges, good as these are for other affections of the fauces. Plenty of food, especially peptonised foods such as Benger's, and alcoholic stimulants, are absolutely necessary. As diarrhœa is not at all a prominent feature of most cases, where the stomach is intolerant of food, or where swallowing is difficult or impossible (where there is the persistent vomiting mentioned and illustrated above), peptonised suppositories and enemas may be employed.

Laryngeal Diphtheria.—As soon as a patient has been discovered to be suffering from this form of diphtheria (whether he or she has been under treatment for diphtheritic pharyngitis or not), a tent should be erected round the bed and a steam-kettle set to work. A few drops of carbolic acid, or some thymol or eucalyptus oil may be put into the kettle; it does little positive good perhaps to have a mild antiseptically-flavoured atmosphere, but it certainly does no harm, and generally pleases the patient's friends if the case is a private one. If the patient is a child, some *vinum ipecacuanhæ* may

be given in two or three full doses to produce vomiting, but should not exceed this number for fear of depressing the system too much. Here it is evident how little practical purpose is answered by much troubling about the distinctions between "membranous croup" and diphtheritic laryngitis—up to this point their treatment is identically the same, and the necessary isolation is nearly equal for both. A lime-water spray should be used for ten minutes or more every three or four hours, and probably the form of spray producer which was popular for surgical operations is the best, as it vaporises the lime-water (although it also dilutes it) more than the ordinary hand spray. Lime-water in the steam-kettle would be excellent, only it will not bear heating. Finally if there be

Membrane on the fauces, Laryngeal stenosis, Recession of chest-walls, and A history of croupy paroxysms	not relieved by one to two hours in a tent and with the steam-kettle,

tracheotomy should be done *at once*. Nothing can be gained by delay; the patient is almost certain to get worse, every hour makes him weaker and less able to stand the shock of the operation, every hour increases the chance of the dreaded spread of the membrane into the lungs. It is a great deal in consequence of tracheotomy having been regarded as the last chance that its successes are not more numerous. *The* mistake which could be made is to diagnose a case of acute laryngitis (with or without membrane) as one of diphtheria; the former might recover without an operation, the latter almost certainly would not. The more I see of diphtheria the more I am convinced that, subject to what has been said above, the earlier tracheotomy is done the better for the patient, and many of the fatal cases recorded in this paper came into the hospital too late, when tracheotomy, though it must be done, becomes indeed a forlorn hope.

Without going into any detail of the operation, I may mention that having tried most of the other kinds, I very much prefer a dilator and ordinary silver tube. And when I was house-

surgeon to Sir Wm. Savory I got Messrs. Ferguson to make me a dilator with a movable catch across the handles, so that when once the tracheal opening has been dilated, and the dilator fixed at the proper width, the mere touch of one finger will keep it in position. This is valuable in such a short-handed operation as an impromptu tracheotomy is and must be, as it allows the operator to get a feather or tube and to introduce it with his right hand while the left holds the dilator—a somewhat troublesome business with the ordinary form, which is always trying to close; or, while the right hand holds the dilator, the tube may be introduced with the left hand, and the right little finger will release the catch of the dilator. The instrument was exhibited this year (1890) at the British Medical Association Meeting at Birmingham.[1]

On one point only in the after treatment of a case of tracheotomy would I venture to lay stress, *i.e.*, that the silver tube be changed for a Baker's rubber one in the first twenty-four or thirty-six hours, and the latter taken out altogether if possible in the first two or three days. The two cases given in the note [2] were

[1] Made and sold by Messrs. Ferguson and Son, West Smithfield, London.

[2] Walter B., aged 5, admitted Nov. 4, 1886. For a week had a husky cold; at 6.40 P.M. had membrane on left tonsil and palate. Recession of chest in Harrison's sulcus, though his colour was fairly good; 8.30, much worse, so Mr. Spencer performed tracheotomy; the child stopped breathing several times and the pulse could not be felt at the wrist. Some membrane was coughed up.

Nov. 5. Silver tube changed for Baker's rubber one. A large piece of membrane expelled.

Nov. 6. Tube taken out altogether. Urine contains albumen.

Nov. 7. More membrane expelled. Patient breathes through his nose.

Nov. 14. Coughs a good deal while feeding.

Nov. 18. Wound is granulating up nicely, the child breathes chiefly through nose and mouth. Urine free from albumen.

Nov. 27. Wound closed, voice quite strong. He is very feeble on his legs, and the patellar reflexes are absent.

He left the hospital on Dec. 15, quite well. He had small doses of iron and nux vomica, and of iron and strychnine all the time he was an in-patient.

Eleanor R., aged 4½, on Nov. 21, 1886, had a "strangling" cough, and was sick. No history of diphtheria near could be gathered. She was admitted at 10 A.M., Nov 26, when she was pallid, with blue lips, metallic voice-sounds, croupy cough, much recession of chest in Harrison's sulcus and in the supra-clavicular and supra-sternal spaces. There was false membrane on fauces and pharynx. Her urine was acid, sp. gr. 1030, and it gave a slight cloud of

instances of the successful accomplishment of this by my friend and colleague, Mr. W. G. Spencer, with the happiest results.

Of intubation of the larynx I have no practical experience worth recording.

albumen. 10.45 Mr. Spencer did tracheotomy, the recession ceased, and in half an hour's time she was asleep. Membrane was coughed up during the operation.

Nov. 27. Silver tube changed for Baker's rubber one.

Nov. 28. Urine more albuminous. Baker's tube removed from 1 P.M. to midnight. A large piece of membrane came away.

Nov. 29. Tube removed altogether.

Dec. 10. Wound closed up.

She went out quite well on Dec. 24. Treatment: iron and nux vomica as in the first case.

DEATHS FROM EXTENSION OF MEMBRANE INTO LUNGS.

Date, &c	Name.—Nature of Disease.	Result.	Age.
1882. M. 50.	J. N——. Membrane on tonsils; tracheotomy performed; membrane coughed up.	Gradually sank.....	7 months.
1882. M. 153.	E. D——. Mar. 9th, croup; membrane on uvula; albuminuria; 14th, tracheotomy; membrane coughed up.	Gradually sank.....	6 years.
1883. M. 713.	S. H——. May 29th, dysphagia; admitted 31st with membrane on fauces, tonsil, and uvula; albuminuria; tracheotomy done same day.	Died after tracheotomy..	2 years.
1883. M. 1420.	J. F——. Breathing bad Dec. 6th; membrane seen on fauces on 10th, and tracheotomy performed.	Dec. 12th, dyspnœa increased, and death occurred.	1 year.
1884. M. 50.	M. C——. Jan. 30th, cough and dyspnœa; admitted 31st; membrane on right tonsil; albuminuria; tracheotomy performed same day. Feb. 1st, membrane expelled; 2nd, cast of trachea brought up.	Died Feb. 3rd. Extension of disease into lungs.	3 years.
1885. M. 71.	A. M. B——. Dec. 26th ('84), glands of neck swollen, and dyspnœa. Jan 1st, admitted with recession dyspnœa, stridor, and aphonia, and membrane on tonsils; tracheotomy; 2nd, pulse irregular; coughed up membrane; duskiness; great albuminuria.	Death Jan. 2nd from extension to bronchi.	2 years 7 months.
1886. M. 168.	Arthur F. L——. Sept. 10th, cough, and later dyspnœa; admitted on 14th; recession, blue colour, and membrane seen on pharynx; urine albuminous; tracheotomy performed; coughed up membrane.	Sept. 16th, died suddenly.	9 months.
1887. M 107.	Nellie S——. July 14th, "clogged at chest"; admitted 16th; drowsy, much membrane on tonsils, croupy cough and voice, stridulous breathing and recession; 17th, tracheotomy; albuminuria; 11 p.m. collapsed, vomiting and diarrhœa.	July 18th, cyanosed occasionally, and collapsed. Died 7.15 p.m.	2 years.
1887. M. 108.	Matilda G——. Aug. 11th, cough; admitted 14th; much recession; copious expectoration; 15th, colour bad; restless; tracheotomy; much false membrane coughed up; albuminuria.	Died Aug. 15th from extension into bronchi.	20 months.

DEATHS FROM EXTENSION OF MEMBRANE INTO LUNGS—*continued.*

Date, &c.	Name.—Nature of Disease.	Result.	Age.
1887. M. 109.	Rebecca W——. Aug. 11th, bad breathing; admitted 12th; stridulous breathing; recession of chest; no membrane seen; tracheotomy; membrane coughed up.	Died Aug. 14th from asphyxia.	18 months.
1887. M. 249.	Walter P——. Dec. 5th, croup began; admitted 7th; recession; no membrane seen; 10th, tracheotomy; respiration bad; no albumen in urine.	Died Dec. 11th. Post-mortem: lung collapsed, and false membrane in larynx and trachea.	18 months.
1887. M. 99.	Clara T——. June 23rd, sore throat, and glands below jaw enlarged; admitted 26th; had two attacks of dyspnœa, 3 and 8 a.m.; pale bluish in colour; much recession; muco-purulent discharge from nose; membrane on left tonsil; and the glands swolleu as above; tracheotomy was performed.	Died June 27th, quietly; no dyspnœa.	4 years 8 months.
1887. M. 123.	Ada W——. July 14th, sore throat; 16th, dyspnœa; 18th, admitted; pale, livid; membrane on fauces; respiration stridulous; recession at base of lungs, and albuminuria; tracheotomy at 6 p.m.; 19th, coughed up membrane; 20th, neck rather swollen; cast of trachea coughed up; 22nd, membrane on bitten lower lip, and on wound; food returned through tube; 23rd, membrane on fauces.	Died July 24th from extension of diseasc on skin and in lungs.	9 years.
1887. M. 199.	Harriet R——. Nov. 27th, cough; 28th, dyspnœa; admitted 30th; livid, with aphonia aud dyspnœa; recession of soft parts of chest; membrane around uvula; tracheotomy was performed.	Died Dec. 2nd..... ...	5 years.
1887. M. 219.	William H——. Dec. 11th, sore throat; 17th, croupy; admitted 18th; bleeding surfaces on tonsils and uvula; glands on left side at angle of jaw enlarged; slight recession; 19th, 5 a.m., tracheotomy; membrane coughed up; urine very albuminous; 20th, large piece of membrane came up in coughing.	Died Dec. 23rd of extensiou into lungs.	5 years.

DEATHS FROM EXTENSION OF MEMBRANE INTO LUNGS—*continued.*

Date, &c.	Name.—Nature of Disease.	Result.	Age.
1887. L. 72.	Charles D——. Mar. 22nd, had a cold, lumps on side of neck, and cough; admitted 26th; pallid; hard and large glands at angle of jaw; membrane on tonsils; voice and cough croupy; respiration stridulous; laryngeal tube introduced.	Mar. 29th, died. Tracheotomy done hastily, and membrane was found to have extended into lungs.	7 years.
1887. L. 172.	David B——. Oct. 25th, languid; 26th, convulsions, lump in neck, breathing bad; admitted 29th; enlarged glands at angle of jaw; membrane on fauces; urine slightly albuminous; 30th, croupy; recession of chest walls; dyspnœa; tracheotomy; membrane coughed up; 31st, more membrane removed. Nov. 1st, a large piece of membrane expelled; urine ¼ albumen.	Died Nov. 2nd from extension of membrane into lungs.	6 years.
1887. L. 229.	Harry D——. July 17th, throat swelled; hoarse; admitted 19th, with membrane on tonsils; glands swollen at angle of jaw; croupy cough; stridulous respiration and recession of the chest; improved under treatment; a trace of albumen in the urine; 23rd, several severe attacks of dyspnœa; 24th, respiration very bad at 5 a.m.; colour blue.	Died July 24th, during tracheotomy.	4½ years.
1882. L. 1312.	Albert F——. June 28th, croupy cough; aphonia; stridulous breathing; 3 p.m. tracheotomy; membrane coughed up; great lividity.	Respiration quickened to death; evident extension of membrane into smaller bronchi.	3 years.
	ONE CASE OF SYNCOPE.		
1887. Feb. 15.	Ernest W——. Feb. 12th, breathing heavy; wheezy; admitted 15th (elder brother ill on the 11th; died 14th of diphtheria); cough; loud breathing; pallid face; running from nose; no membrane seen on fauces; cough rather whoopy; recession in Harrison's sulcus; 16th, no membrane seen; neck swollen; voice sounds metallic; dyspnœa in night.	Feb. 17th, went off suddenly to-day; not much dyspnœa.	13 months.

Three Cases of Death from Bronchitis in Diphtheria.			
Date, &c.	Name.—Nature of Disease.	Result.	Age.
1881. M. 398.	E. M——. Dysphagia Sept. 2nd; tracheotomy was performed on the 5th; no membrane had been seen, but some was coughed up; there was no albuminuria.	Sank gradually, bringing up a great deal of mucous expectoration.	2 years.
1883. M. 623.	M. A. R——. Cough June 2nd; admitted 4th; urine albuminous; tracheotomy on the 5th; membrane expelled afterwards.	An attack of bronchitis came on and killed the child.	18 months.
1889. M. 165.	Sarah B——. Vomiting Oct. 2nd; and dyspnœa on the 5th; admitted on the 6th with stridulous breathing; livid colour; much recession of chest, and mucous *râles* at base of chest.	Died, asphyxiated from the bronchitis, on Jan. 7th.	15 months.
Four Fatal Cases of Diphtheritic Laryngitis.			
1884. M. 717.	A. F——. Dyspnœa May 24th; admitted 25th with stridulous dry cough; no membrane visible; great recession of chest.	Died on table during tracheotomy, May 25th.	2 years.
1885. M. 659.	Eliza G——. Sore throat June 16th; admitted on the 20th with muco-purulent discharge from nostrils; membrane on tonsils and soft palate; stridulous respiration; some recession of chest; urine ½ albumen.	Died June 21st (not distinctly asphyxiated).	1 year 8 months.
1887. M. 187.	Thomas J——. Some dyspnœa Nov. 4th; admitted on the 5th with slightly enlarged glands at the angles of jaw, croupy cough, and some recession of chest; no membrane was seen; no improvement in six hours, so tracheotomy was performed.	Died two days after tracheotomy.	9 months.
1884. L. 251.	David S——. Jan. 30th ill; admitted Feb. 2nd with cough, free expectoration; dysphagia; epiglottis swollen, dusky red; and the front of his neck swollen.	Died asphyxiated, Feb. 3rd. Post-mortem: Cellulitis of front of neck; œdema of ary-epiglottic folds, and there was diphtheritic membrane on those folds.	46 years.